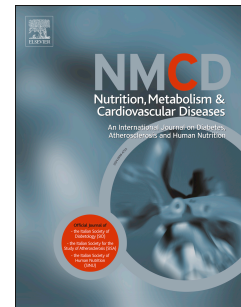


Accepted Manuscript

A pro-inflammatory diet is associated with increased risk of developing hypertension among middle-aged women

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PII: S0939-4753(17)30061-3

DOI: [10.1016/j.numecd.2017.03.005](https://doi.org/10.1016/j.numecd.2017.03.005)

Reference: NUMECD 1709

To appear in: *Nutrition, Metabolism and Cardiovascular Diseases*

Received Date: 23 November 2016

Revised Date: 15 March 2017

Accepted Date: 16 March 2017

Please cite this article as: Vissers L, Waller M, van der Schouw Y, Hébert J, Shivappa N, Schoenaker D, Mishra G, A pro-inflammatory diet is associated with increased risk of developing hypertension among middle-aged women, *Nutrition, Metabolism and Cardiovascular Diseases* (2017), doi: 10.1016/j.numecd.2017.03.005.

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Dietary Inflammatory Index and hypertension.

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Abstract: 199 words

Manuscript: 3,125 words

Keywords: Hypertension, Diet, inflammation

Abstract

Background and Aims– A pro-inflammatory diet is thought to lead to hypertension through oxidative stress and vessel wall inflammation. We therefore investigated the association between the dietary inflammatory index (DII) and developing hypertension in a population-based cohort of middle-aged women.

Methods and Results – The Australian Longitudinal Study on Women’s Health included 7,169 Australian women, aged 52 years (SD 1 year) at baseline in 2001, who were followed up through 4 surveys until 2013. The DII, a literature-derived dietary index that has been validated against several inflammatory markers, was calculated based on data collected via a validated food-frequency questionnaire administered at baseline. Hypertension was defined as new onset of doctor-diagnosed hypertension, ascertained through self-report between 2001 and 2013. Generalised Estimating Equation analyses were used to investigate the association between the DII and incident hypertension. The analyses were adjusted for demographic and hypertension risk factors. During 12-years follow-up we identified 1680 incident cases of hypertension. A more pro-inflammatory diet was associated with higher risk of hypertension in dichotomised analyses with an $OR_{\text{fully adjusted}}$ of 1.24, 95% CI: 1.06-1.45.

Conclusion – A pro-inflammatory diet might lead to a higher risk of developing hypertension. These results need to be replicated in other studies.

List of abbreviations

ALSWH = Australian Longitudinal Study on Women's Health, BMI = Body Mass Index, CI = confidence interval, DII = Dietary Inflammatory Index, FFQ = Food Frequency Questionnaire, g = gram, GEE = Generalised Estimating Equation, kJ = kilojoule, kcal = kilocalorie, mg = milligram, MUFA = monounsaturated fatty acids, OR = odds ratio, PUFA = polyunsaturated fatty acids, SD = standard deviation. μg = microgram

Introduction

There are several dietary patterns that have been shown to decrease risk of hypertension, such as the Mediterranean Diet Score (MDS) [1] and the Dietary Approaches to Stop Hypertension (DASH) diet [2]. These type of diets describe specific beneficial or detrimental food items that are culturally defined (e.g. olive oil in the Mediterranean diet) or empirically derived (e.g. less red meat in the DASH diet) [1, 2]. Although the associations between adherence to these diets and decreased hypertension risk are clear [1, 2], adherence to these diets might be difficult in populations with other cultural traditions and dietary habits [3].

A different approach, aimed at metabolic function, is to assess the properties of an existing dietary pattern as done by the dietary inflammatory index (DII) [4, 5]. The DII has the capacity to describe inflammatory capacity across culinary traditions. In time, this could be used to make healthy suggestions within each person's (often culturally defined) diet, in order to provide tailored dietary advice. The added value of this new dietary score over existing patterns is therefore applicability to different populations. The DII has been developed based on an extensive literature review through 2010 [4, 5]. Food parameters were categorised as pro- or anti-inflammatory or inflammatory neutral to calculate a total inflammatory score for the overall diet[5]. The relationship between the DII and inflammatory markers and health outcomes has been demonstrated previously [6].

Among other mechanisms, inflammation can have a detrimental effect on the vascular system and kidney function, which in turn can lead to hypertension [7].

The relationship between the DII and hypertension has been investigated previously in cross-sectional studies with population based cohorts of men and women[8, 9]. One of these studies, performed in an American cohort, observed an association between pro-inflammatory DII scores and higher risk of hypertension [9]. The aim of this study is to prospectively investigate the association between the DII and incident hypertension in a population-based cohort of middle-aged women.

Methods

Study population

The Australian Longitudinal Study on Women's Health (ALSWH) recruited a nationally representative sample of over 40,000 women in 1996 [10]. Women were sampled from the National health insurance scheme (Medicare) that includes all Australian citizens and permanent residents. Three age groups were sampled; women born in 1973-78, 1946-51, and 1921-26. Sampling of women was random, except that women from rural and remote areas were intentionally oversampled [10].

For this study we used the cohort of women born in 1946-51. This cohort of middle-aged women has been surveyed every 2-3 years since the start of the ALSWH in 1996, with a total follow up of 17 years. Based on the initial response of 13,715 to Survey 1, response rates for Surveys 2, 3, 4, 5, 6 and 7 were $n = 12,338$ (90%), $n = 11,221$ (81.8%), $n = 10,905$ (79.5%), $n = 10,638$ (77.6%), $n = 10,011$ (73.0%) and $n = 9,151$ (66.7%), respectively [11]. Dietary information was assessed at Survey 3 in 2001 and this survey was therefore used as baseline for our study. Participants with missing nutritional data were excluded ($n=593$). Furthermore, women with hypertension at baseline were excluded ($n=3,056$), as were women with missing information on hypertension at baseline ($n=403$), leaving 7,169 women for data analysis.

The Human Research Ethics Committees of the University of Newcastle and the University of Queensland approved the study methods. Further details on the sample and methods used by the ALSWH have been reported elsewhere [10, 12] and are available at www.alswh.org.au.

Ascertainment of hypertension

Self-reported data on doctor-diagnosed hypertension was available from each survey. At Survey 1, participants were asked if a doctor ever diagnosed them with hypertension. At all subsequent surveys participants were asked if they had been diagnosed with hypertension by a doctor in the last 2-3 years, coinciding with the time since last survey. If women reported presence of hypertension at Survey 1, 2 or 3, they were excluded from analysis. Incidence of hypertension was defined as new onset of hypertension at Survey 4 to 7. A study in an ALSWH sub-cohort investigated self-reported survey information for hypertension and compared this to hospital-derived data [13]. For the 1946-1951 cohort, hospital data between 2004 and 2008 was used and estimated hypertension prevalence to be 12.8% (95%CI 10.8 to 14.8%). In response to surveys, however, 45.4% (95%CI 42.4 to 48.4%) of women had ever indicated having hypertension [13]. The latter estimate is similar to the expected hypertension prevalence among women aged 50-59 in established market economies of approximately 42% [14]. Furthermore, we examined the agreement between self-reported hypertension and medication use in our study population at Survey 4, which was high (89%).

Dietary intake

Information on dietary intake over the past 12 months was obtained from a food frequency questionnaire (FFQ), The Dietary Questionnaire for Epidemiological Studies, version 2 (DQES v2) [15], which has been validated against seven days of weighed food records, showing a Pearson correlation of 0.32-0.70 for intake of nutrients (elog transformed and energy adjusted) that were included in the DII [16]. Participants were asked to estimate portion sizes and report their usual daily frequency of consumption of 101 individual food items on a 10-point scale ranging

from 'Never' to 'Three or more times per day' [15]. The Australian Food Composition Database (NUTTAB95) was used to calculate energy and nutrient intakes [17].

Dietary Inflammatory Index

The development and validation of the DII are described in detail elsewhere [4]. Developing the DII involved reviewing and scoring nearly 2000 scientific articles representing cell culture and laboratory animal experiments, and a variety of human studies on diet and six inflammatory markers (i.e., CRP, interleukin (IL)-1b, IL-4, IL-6, IL-10, tumour necrosis factor (TNF)- α).

Developing the DII also entailed creation of a world standard database that involved obtaining 11 data sets from around the world to which individuals' intakes of 45 food parameters (consisting of nutrients, spices and whole foods) on which the DII is based, could then be compared.

FFQ-derived dietary data were used to calculate DII scores for all participants. Dietary data were first linked to the previously described regionally representative world database that provided a robust estimate of a mean and standard deviation for each parameter [4]. These then became the multipliers to express an individual's exposure relative to the "standard global mean" as a z-score. This score was computed by subtracting the "standard global mean" from the amount reported and dividing this value by the "global standard deviation" of the world population as represented by the 11 data sets used for comparative purposes. To minimize the effect of "right skewing," which commonly occurs for dietary variables, this value was then converted to a centered percentile score.

For each individual food parameter, this score was multiplied by the respective food parameter effect score, derived from the literature review, in order to obtain a food parameter-specific DII score [4]. All of the food parameter-specific DII scores were then summed to create the overall DII score for each participant in the study, $DII = b_1 * n_1 + b_2 * n_2 + \dots + b_{29} * n_{29}$, where b refers to the literature-derived inflammatory effects score for each of the evaluable food parameters and n refers to the food parameter-specific centered percentiles, which were computed from the FFQ-derived dietary data. The DII has been associated with higher levels of inflammatory markers in multiple cohorts[5, 18-20].

For the current study, data on 25 of the 45 DII food parameters could be derived from the FFQ and were thus used for DII calculation. These include: Pro-inflammatory components (energy, carbohydrate, protein, fat, saturated fat, iron, cholesterol) and anti-inflammatory components (alcohol, fibre, mono-unsaturated fat, poly-unsaturated fat, omega-3, omega-6, niacin, thiamin, riboflavin, magnesium, zinc, vitamin A, vitamin C, vitamin E, folic acid, beta-carotene, garlic and onions). We did not have FFQ data on intake of Vitamin B12, Vitamin B6, caffeine, eugenol, ginger, saffron, selenium, *trans* fat, turmeric, vitamin D, green/black tea, flavan-3-ol, flavones, flavonols, flavonones, anthocyanidins, isoflavones, pepper, thyme/oregano or rosemary. The total DII score has been dichotomised into generally pro-inflammatory ($DII \geq 0$) and generally anti-inflammatory ($DII < 0$) to compare the additive effect of all these factors. Theoretically, the DII can range from the most pro-inflammatory score of +7.98 to a most anti-inflammatory score of -8.87.

Covariates

At every survey; participants were asked to provide information about a range of demographic and socio-economic factors and health risk behaviours, including level of education (low – no formal qualifications or school or intermediate certificate or equivalent; intermediate – high school or leaving certificate, trade/apprenticeships, or certificate or diploma; or high – university degree); smoking (non-smoker, former smoker or current smoker); and doctor-diagnosed diabetes. Menopause status was determined using questions on hysterectomy, oophorectomy, hormone therapy and menstrual pattern, and categorised as ‘hysterectomy and/or oophorectomy’, ‘hormone therapy use’, ‘pre-menopausal’, ‘peri-menopausal’ or ‘post-menopausal’[21]. Participants were asked about their height and weight. BMI was calculated and categorised as underweight (BMI <20 kg/m²), normal weight (BMI 20-25 kg/m²), overweight (BMI 25-30 kg/m²) and obese (BMI >30 kg/m²)[22]. Physical activity scores were derived from validated questions on frequency and duration of walking (for recreation or transport) and from moderate- and vigorous-intensity activity in the last week and categorised as: ‘sedentary/low’ (<600 total metabolic equivalent (MET) minutes/week) or ‘moderate/high’ (≥600MET minutes/week)[23].

Data analysis

We analysed the prospective association between the DII (Survey 3) and incidence of self-reported hypertension (Survey 4-7), using Generalised Estimating Equations (GEE) analysis. An unstructured correlation structure was used to take into account the repeated measurements for each subject in this longitudinal dataset. Data for women who died during follow up or withdrew from the study were included in the analysis up to the point of censoring.

After testing for non-linearity, we assessed the association with hypertension for DII as a continuous variable per 1 standard deviation (SD) increase in score. Furthermore, we investigated the effect of a pro-inflammatory diet in dichotomised analyses (DII<0 versus DII≥0). Models were adjusted for time-varying confounders (Survey 3-6). Several confounders were defined *a priori* based on literature such as age, smoking, diabetes, menopausal status, physical activity and BMI. We did not correct for alcohol intake, since alcohol is a part of the DII. Education was selected based on statistical significance in the model. The first model was adjusted for energy intake and survey number. In the second model we additionally adjusted for hypertension risk factors and demographic characteristics. We corrected for BMI in a third model, because this also might be an effect modifier.

To assess the impact of missing survey data, we performed multiple imputation (n=20) at baseline and carried the last known value forward. Given the limitations of this type of imputation for longitudinal data [24], imputed results are presented as sensitivity analyses. All analyses were performed using SAS® Software version 9.4 (SAS Institute Inc., Cary, NC, USA).

Although diabetes and high BMI might partially represent the same increased risk of hypertension, we also expect that type 2 diabetes might exert an independent effect because it is under considerable genetic control [25]. We were concerned that the effect of a pro-inflammatory diet might be different for women with diabetes or women in a high BMI group at baseline. We have therefore investigated effect modification by including the interaction term (exposure x covariate) in model 3.

Results

At baseline, the 7,169 women included in this study had a mean age of 52 years (SD 1 year).

There were 1,664 women with a mainly anti-inflammatory diet, whereas 5,505 women had a more pro-inflammatory diet. A pro-inflammatory diet was observed in women who had lower prevalence of diabetes, but were more likely to smoke, be in a high-risk drinking category and be physically inactive (Table 1).

The pro-inflammatory diet was lower in energy intake (5900 kJ/day versus 9010 kJ/day) and absolute intake of all individual nutrients (Table 2). Nutrient densities (intake of nutrient per 1000 kcal) of total fat, saturated fat and calcium were higher in women consuming the more pro-inflammatory diet, but concentrations of all other nutrients were lower (data not shown). Also, women with a pro-inflammatory diet had a lower intake of all food groups, especially fish, vegetables, fruit, nuts, potatoes, pasta and rice. Only intake of high-fat dairy was suggested to be higher with a pro-inflammatory diet (Table 2).

During 12 years of follow-up we registered 1680 new cases of self-reported hypertension.

A more pro-inflammatory diet, according to the DII, was significantly associated with a higher risk of incident hypertension after multivariable adjustment (model 3) in comparison to the anti-inflammatory diet, with a 24% (95% CI: 6 to 46%) higher risk. Results for continuous analyses were borderline significant and suggested a 7% (95% CI: -1 to 15%) higher risk of hypertension (p-value 0.08) per 1 SD increase in DII score. Sensitivity analysis with imputed data did not change observed associations (data not shown).

No interaction was found between DII and diabetes (p-value for interaction = 0.84) or DII and BMI risk group (p-value for interaction = 0.56).

Discussion

Among middle-aged Australian women, we found an association between a more pro-inflammatory diet and higher risk of developing hypertension during 12 years of follow up. A pro-inflammatory diet led to a 24% higher risk of hypertension when compared to an anti-inflammatory diet.

The pro-inflammatory diet was characterised by low intake of fish, vegetables, fruit, nuts, potatoes, pasta and rice and a high intake of high-fat dairy. Total energy intake (a pro-inflammatory DII component) was lower in the group with a pro-inflammatory diet, which is probably caused by the lower intake of the abovementioned whole foods with a high nutrient density. Many of these nutrients are characterised as anti-inflammatory. The higher intake of other pro-inflammatory DII constituents (e.g. saturated fat or protein) in the anti-inflammatory is a result of the DII algorithm – All individual parameter scores of the diet are summed to reflect a net effect of this diet on inflammation, rather than focussing on individual nutrients. Characterising a diet based on its inflammatory properties is also performed by the Adapted Dietary Inflammatory Index [26], but this index has not been investigated in relation to hypertension. The anti-inflammatory diet found in this cohort seems comparable to many existing dietary patterns that are recommended for CVD prevention with respect to specific food intakes (high intake of fish, vegetables, fruit and nuts)[1, 2]. This is consistent with correlations observed between DII scores and other dietary indices such as the MDS, DASH, the Recommendation Compliance Index and the Diet Quality Index, among others[8]. It would be interesting to compare the different indices with regard to their effect on inflammation and hypertension.

The DII has been investigated previously in the ALSWH cohort with regard to CVD, but no association was observed [27]. An association between a pro-inflammatory diet and hypertension incidence in the same cohort might seem surprising because hypertension is one of the main risk factors for CVD. However, wide confidence intervals for the association between the DII and CVD subtypes suggest that there might have been a lack of power rather than a lack of effect [27]. Other studies did find a relationship between the DII and CVD (risk factors) [6]. The observed relationship between the DII and risk of hypertension in the ALSWH cohort is in line with results from a previous population-based cross-sectional study [9]. The observed relationship between the DII and hypertension is in line with our expectations as the DII is constructed from nutrients and whole foods that have been related to inflammatory markers [4, 5], which in turn have been related to hypertension[7]. A meta-analysis has provided further evidence for a causal relationship between inflammation and CVD - genetic variants that impair classic IL-6 signalling in the pro-inflammatory cascade have been associated with lower levels of CRP (and other acute-phase reactants) and to a lower risk of CHD [29]. The lower prevalence of diabetes and lower BMI at baseline in participants with a pro-inflammatory diet is not in line with previous studies that observed no difference in risk or a higher risk of metabolic syndrome in participants with a more pro-inflammatory diet [6]. Given the age of the participants in this study, it is possible that women with diabetes or a high BMI have already adjusted their dietary patterns after receiving nutritional counselling from health care professionals before inclusion in this study.

Strengths of this study include the long duration of follow up with repeated survey data of a population based sample of Australian women and exploration of the influence of missing data

through multiple imputation. Despite its strengths, there are some study limitations worth noting. First of all, we cannot exclude the possibility that the diet measured at baseline was adopted only shortly before administration of the FFQ although the prospective nature of the study minimizes problems resulting from potential reverse causation. Secondly, women with a pro-inflammatory diet tended to have a less healthy lifestyle at baseline compared to women with an anti-inflammatory diet. Even though we simultaneously corrected for multiple demographic and hypertension risk factors, we cannot exclude residual confounding due to imprecisely measured or unmeasured factors. We did not have information on medication use at baseline so we were unable to investigate presence of an interaction between the DII and medication that could modulate inflammatory responses, such as statins [28]. Also, hypertension was present in almost 30% of the otherwise eligible ALSWH cohort at baseline and these women were excluded from data analysis. Therefore, results require confirmation in different cohorts before being generalised to a younger population. Thirdly, dietary intake was assessed only at baseline and might not represent dietary habits well over a period of 12 years. A German study showed that dietary pattern classification is moderately stable over a 6-year period in their sub-cohort of women aged 35-65 years [29], but we cannot exclude the possibility of a change in dietary habits during our follow-up. Also, our study is limited by the fact that only 25 out of 45 food parameters were available. However, the DII has been validated using a variety of inflammatory markers in multiple other cohorts [6] including those where fewer than 30 DII parameters were available [18-20]. The use of self-reported data for ascertainment of hypertension is a possible limitation although we did observe a high correlation between self-reported doctor-diagnosed hypertension and use of antihypertensive

medication in our study. Finally, as in any observational study, we are faced with the problem of loss to follow up. However, it is highly unlikely that a person is unable or unwilling to fill in the next survey as a result of being diagnosed with hypertension, so we do not expect this loss to follow up to be selective.

Conclusion

We report a positive association between a more pro-inflammatory diet and development of hypertension during 12 years of following this cohort of middle-age women. If our results can be confirmed in other cohorts, culturally appropriate dietary advice to prevent hypertension could be provided.

Acknowledgements

The research on which this paper is based was conducted as part of the Australian Longitudinal Study on Women's Health, the University of Newcastle and the University of Queensland. We are grateful to the Australian Government Department of Health for funding and to the women who provided the survey data.

The authors thank Professor Graham Giles of the Cancer Epidemiology Centre of The Cancer Council Victoria, for permission to use the Dietary Questionnaire for Epidemiological Studies (Version 2). Melbourne: The Cancer Council Victoria, 1996.

Funding

Drs. Shivappa and Hébert were supported by grant number R44DK103377 from the United States National Institute of Diabetes and Digestive and Kidney Diseases. Prof. Mishra is supported by an Australian Research Council Future Fellowship (FT120100812)

Disclosure footnote

Dr. James R. Hébert owns controlling interest in Connecting Health Innovations LLC (CHI), a company planning to license the right to his invention of the dietary inflammatory index (DII) from the University of South Carolina in order to develop computer and smart phone applications for patient counseling and dietary intervention in clinical settings. Dr. Nitin Shivappa is an employee of CHI.

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Table 1. Baseline description by anti- inflammatory and pro-inflammatory diet, Australian Longitudinal Study on Women's Health, 1996 to 2013, N = 7169.

	Anti- Inflammatory	Pro- Inflammatory	p-value
Women (n)	1664	5505	
Dietary Inflammatory Index score	< 0	>0	
Dietary Inflammatory Index	-1.05 ± 0.81	1.86 ± 1.01	<0.0001
Age (years)	53 ± 1	52 ± 1	<0.0001
Height (cm)	163.8 ± 6.6	162.9 ± 6.6	<0.0001
Weight (kg)	73.1 ± 15.7	70.8 ± 14.7	<0.0001
BMI (kg/m ²)	27.2 ± 5.7	26.7 ± 5.4	0.0007
Urban area of residence (%)	36.1	39.4	0.02
Highly educated (%)*	17.4	15.7	0.06
Overweight or obese (%)	51.6	48.5	0.08
Smoking (%) Current	9.4	16.6	<0.0001
Former	23.8	24.2	<0.0001
Physically inactive (%)†	47.2	54.8	<0.0001
Type 1 or 2 diabetes (%)	4.2	2.9	0.002
Stroke (%)	0.4	0.7	0.25
Heart Disease (%)	1.0	1.1	0.65
Post-menopausal (%)	25.1	25.9	0.23
Hormone therapy use (%)	15.5	17.2	0.23

Data are percentages (%), means ± SD or median (interquartile range). SD = standard deviation. P-value represents level of significance for differences found through t-test (continuous variables) or chi-square test (categorical variables). For categorical variables the difference between variable upon which percentages are based is noted (variables described in covariate method section). * Any university schooling † sedentary or low activity, i.e. <600 Metabolic equivalents per week.

Table 2. Dietary description anti- inflammatory and pro-inflammatory diet, Australian Longitudinal Study on Women's Health, 1996 to 2013, N = 7169.

	Anti- Inflammatory	Pro- Inflammatory	p-value
Energy (kJ/day)	9010 ± 3020	5900 ± 1750	<0.0001
Glycaemic index *	51.5 ± 3.7	52.0 ± 4.4	<0.0001
Total fat (g/day)	82.9 ± 37.2	56.5 ± 21.4	<0.0001
Saturated fat (g/day)	31.4 ± 16.5	23.1 ± 10.3	<0.0001
PUFA (g/day)	14.1 ± 6.1	8.5 ± 3.9	<0.0001
MUFA (g/day)	29.7 ± 14.2	19.8 ± 7.7	<0.0001
Protein (g/day)	111 ± 46	72.1 ± 23.6	<0.0001
Carbohydrates (g/day)	243 ± 72	155 ± 47	<0.0001
Sugars (g/day)	109 ± 34	72 ± 24	<0.0001
Fibre (g/day)	30.5 ± 8.1	17.0 ± 5.1	<0.0001
Calcium (mg/day)	1055 ± 344	789 ± 276	<0.0001
Iron (mg/day)	17.3 ± 6.1	9.8 ± 3.4	<0.0001
Zinc (mg/day)	14.6 ± 5.8	9.4 ± 3.3	<0.0001
Magnesium (mg/day)	384 ± 105	228 ± 63	<0.0001
Phosphorus (mg/day)	1900 ± 610	1230 ± 360	<0.0001
Sodium (mg/day)	2870 ± 1150	1880 ± 615	<0.0001
Potassium (mg/day)	3650 ± 900	2300 ± 600	<0.0001
RE (µg/day) •	1000 ± 320	641 ± 222	<0.0001
Vitamin C (mg/day)	177 ± 79	100 ± 49	<0.0001
Vitamin E (mg/day)	8.2 ± 2.5	4.8 ± 1.5	<0.0001
High fat dairy (g/day)	8 (28.3)	12.0 (200.6)	<0.0001
Low fat dairy(g/day)	375 (282)	220 (342)	<0.0001
Total meat (g/day) ¥	158 ± 130	105 ± 69	<0.001
Red meat (g/day) ¥	56.1 ± 55.2	35.9 ± 33.9	<0.001
Fish (g/day) ¥	55.5 ± 74.2	27.7 ± 26.5	<0.001
Eggs (g/day)	12.9 (6)	12.9 (6)	<0.0001
Potatoes (g/day)	51.4 ± 39.7	29.9 ± 27.6	<0.001
Pasta (g/day)	57.0 ± 53.3	34.1 ± 30.7	<0.001
Rice (g/day)	50.1 ± 66.0	28.1 ± 37.4	<0.001
Vegetables (g/day) ¥	181 ± 71	107 ± 47	<0.001

Legumes (g/day) ¥	23.0 ± 17.1	15.4 ± 12.1	<0.001
Fruit (g/day) ¥	315 ± 160	179 ± 115	<0.001
Nuts (g/day) ¥	8.7 ± 12.0	3.4 ± 4.9	<0.001
Sweets (g/day) ¥	52.6 ± 60.3	31.9 ± 34.6	<0.001
Savoury snacks (g/day) ¥	72.5 ± 85.3	50.8 ± 38.2	<0.001
Alcohol (g/day)	9.6 ± 13.7	9.4 ± 13.0	0.51

Data are means ± SD or median (interquartile range). P-value represents level of significance for differences found through t-test (continuous variables) or chi-square test (categorical variables). SD = standard deviation, PUFA = polyunsaturated fat MUFA = monounsaturated fat * Average ranking of carbohydrates on a scale from 0 to 100 according to the extent to which they raise blood sugar levels after eating within total diet. • 1µg of retinol equivalent equals 1 µg of all-trans retinol, 6 µg all-trans beta-carotene, 12 µg of α-carotene, beta-cryptoxanthin and other provitamin A carotenoids ¥ Calculated by adding grams/day intake of several food items (High fat dairy: Cream, soft, firm and hard cheese + full cream milk and flavoured milk drink. Low fat dairy: Low fat cheese, ricotta or cottage cheese, reduced fat, skim or soya milk, yoghurt. Total meat: Beef, chicken, ham, hamburger, lam, pork, salami, sausages, veal. Red meat: Beef. Processed meat: Ham, salami, sausages. Fish: Fish and fried/tinned fish. Vegetables: Avocado, beetroot, broccoli, cabbage, capsicum, carrots, cauliflower, celery, garlic, lettuce, mushrooms, onion, peas, pumpkin, spinach, tomatoes, zucchini. Legumes: Baked beans, bean sprouts, green beans, other beans, tofu. Fruit: Apples, apricots, bananas, mango, melon, oranges, peaches, pears, pineapple, strawberries. Nuts: Nuts and peanut butter. Sweets: Cakes, chocolate, ice cream, sweet biscuits. Savoury snacks: Chips, crisps, fried fish, hamburger, meat pies, pizza, salami, sausages)

Table 3. Dietary inflammatory index and risk of incident hypertension among 7,169 middle-age**Australian women, Australian Longitudinal Study on Women's Health, 1996 to 2013.**

Dietary Inflammatory index	Anti-Inflammatory*	Pro-Inflammatory*	per 1SD increase in DII
Women (n)	1664	5505	
hypertension, n(%)	380 (22,8%)	1300 (23,6%)	
Dietary Inflammatory Index Score	-1.05 ± 0.81	1.86 ± 1,01	
Time and energy adjusted	1	1.17(1.02-1.35)	1.08(1.01-1.16), p value 0.02
Model 2 †	1	1.19(1.02-1.38)	1.06(0.98-1.13), p value 0.15
Model 3 ‡	1	1.24(1.06-1.45)	1.07(0.99-1.15), p value 0.08

Data are ORs (95% CI). DII is expressed as median (interquartile range). OR = odds ratio, CI = confidence interval, SD = standard

deviation, DII = Dietary Inflammatory Index. *Anti-inflammatory = index score <0, used as reference. Pro-inflammatory = index

score ≥ 0 † Adjusted for time and energy + age, diabetes, smoking status, education, menopausal status and physical activity. ‡

Adjusted for model 2 + for BMI.

Highlights

- A pro-inflammatory diet is associated with higher hypertension incidence in women.
- The pro-inflammatory diet was characterised by low intake of fish, vegetables, fruit, nuts, potatoes, pasta and rice and high intake of high-fat dairy.
- No interaction between inflammatory potential of diet and BMI or diabetes was found.